

Etiology of Femoral Hernia

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Introduction

DESPITE the voluminous literature on the subject of the etiology of the femoral hernia, there is no unanimity of opinion. The theory that there is a congenital pre-formed peritoneal sac is untenable to us but through the years various authors have advanced this as the principal etiology in the development of the femoral hernia. Murray,⁸ in 1910, favored the saccular theory and cited his observations on 200 consecutive autopsies in which 47 had femoral diverticula. He further postulated that the female pelvis expands laterally at puberty and this tends to widen the mouth of the femoral diverticulum; that this coupled with increased intra-abdominal pressure, such as with pregnancy, combines to cause a femoral hernia. As recently as 1923 Russell¹⁰ still favored the congenital peritoneal sac theory.

The observations of the senior author of this paper on the dissection of more than 600 cadavers fails to substantiate the peritoneal sac theory. Although a peritoneal dimple is frequently found over the femoral ring in an emaciated cadaver, this is not deep enough to be designated a peritoneal diverticulum or hernial sac. The only instance of a congenital peritoneal sac through the femoral ring known to these authors is the report of Fauntleroy,² in 1920, in which the testicle had descended through the femoral ring into the scrotum and the patient concomitantly also had an indirect inguinal hernia which of necessity

then passed through the femoral ring. Wood,¹³ in 1906, collected 100 cases from the literature in which an appendix was found in a femoral hernial sac and he believed that the hernial sac in a femoral hernia could be either congenital or acquired.

In 1923, Keith³ stated that there is never an evagination of peritoneum through the femoral ring in either man or beast and he believes that the femoral ring represents a safety valve for expansion of the femoral vein in the upright posture but at the same time this provides a potential space for a hernia. He stated that any straining effort causes an impulse in the femoral vein which strikes a blow at the femoral ring and eventually causes a dilatation of the ring. Preperitoneal fat may then be forced as a wedge into the traumatized and enlarged femoral ring causing a hernia. Keith completely discredited the theory that an individual who does not have a congenital hernial sac will never have a hernia. He emphasized that any weak area in the abdominal wall subjected to a constant repetition of increased intra-abdominal pressures will develop a hernia at that site. Referring to his knowledge of embryology and anatomy he emphasized that congenital pockets are not a necessity and that all hernias result from weakened areas of the abdominal wall with the repetitive episodes of abdominal trauma being the exciting cause. He further believed that even if a sac were present it was not the most important factor in the development of even the indirect groin hernia.

* By invitation.

Buckley,¹ in 1924, stated that the femoral hernia was acquired and that fat preceded and pulled the peritoneum with it. He further stated that childbearing was the only difference in the sexes and that the incidence was the same prior to parturition.

Tasche,¹¹ in 1932, thought that the hernial sac was never congenital but acquired and produced by traction, not pressure. In his experience the female has twice as many femoral hernias as the male but the male has just as many asymptomatic peritoneal diverticula at the femoral ring as the female. Tasche made many anatomical studies and measurements and stated that the lacuna vasorum increases in size in both sexes from fetal life to old age and that this enlargement favors the production of a femoral hernia because it allows the force of traction to act to better advantage. He also observed that the lacuna vasorum is smaller in the female and that Poupart's ligament is likewise smaller in the female. He also stated that the femoral artery and vein increase in size with age. He further stated that the available space for the development of a femoral hernia is smaller in the female than in the male, or in other words, the difference between the sum of the areas of the two femoral vessels subtracted from the total of the lacuna vasorum is greater in males than it is in females.

Thomas,¹² in 1915, was not concerned with a preformed peritoneal diverticulum as with an inguinal hernia but thought that a ring existed as with the umbilical hernia and that the development of a hernial sac was secondary to pressures against this ring.

In 1922, Panton⁹ based his theory upon the evolution of man into the upright position and the plantigrade mode of progression as being etiologic. This has resulted then in the opening of the foot, leg and thigh in a manner that is quite different from the lower or hind extremity of even the apes. He believed that this "opening

up" has reached up to the groin causing an enlargement of the communication between groin and abdomen, and that this necessarily then means a weakening of this portion of the parieties of the abdomen. Therefore, because of the normally erect trunk, directly over a normally extended lower limb, the weight of the abdominal contents and the force of gravity all then act directly and he stated, "herein then lies the secret of the human predisposition to femoral hernia." He also believed in the presence of a congenital peritoneal diverticulum. His paper also contains many measurements of the pelvocrural interval and the length of Poupart's ligament. We have made these measurements too but do not believe there is enough difference in the two sexes to be of any significance. He does point out that the female false pelvis is either relatively narrower than the male or else is equal to it and therefore the wider pelvis theory of femoral hernia is untrue and in this we concur.

The references above are representative of the many papers that have been written regarding the etiology of the femoral hernia. Although we are in complete agreement with those authors who state that the femoral hernia is an acquired hernia, the study of the literature still leaves one in a quandry as to the exact etiology of the femoral hernia. While the femoral hernia is most common in the multiparous female, most multiparous females of course, do not develop a femoral hernia.

After studying the problem one is left with the inescapable and trite conclusion that the fundamental etiology of a femoral hernia is an enlarged femoral ring. However, the factors that cause the femoral ring to enlarge are more obscure. Through the years we have noted considerable variation in anatomic structure about the femoral ring and considerable variation in the size of the femoral ring at the operating table when repairing inguinal hernias. It

is with these impressions in mind that the present study was undertaken.

Materials and Methods

In routine cadaver dissections, and while making other measurements in the inguino-femoral region, we have noted considerable variation in the breadth of the structures that attach to Cooper's ligament and in the transverse diameter of the femoral ring. Many years ago one of us (C. B. M.)⁴ measured the breadth of the attachment of the

posterior inguinal wall onto Cooper's ligament in a few cadavers and made the notation that there is considerable variation in the breadth of this attachment and that there is a rough correlation between the breadth of this attachment and the transverse diameter of the femoral ring. That is to say that the broader the posterior inguinal wall attachment onto Cooper's ligament, the narrower the transverse diameter of the femoral ring, and that the converse is also true. With this in mind and with

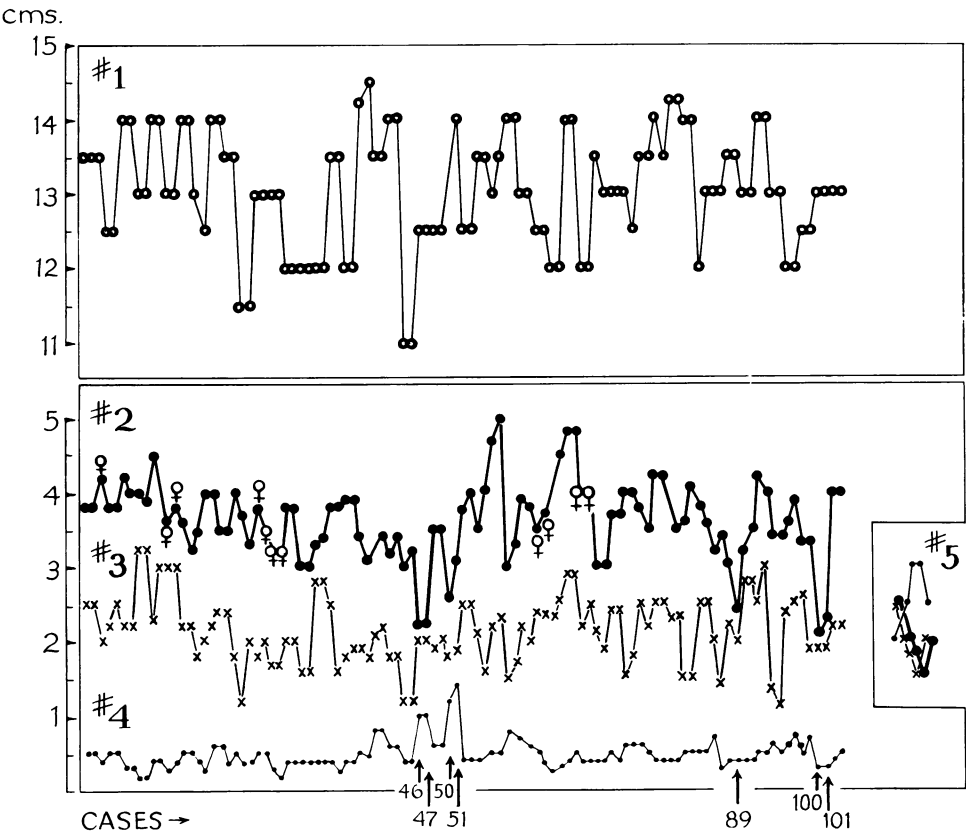


FIG. 1. These graphs represent measurements made upon 103 cadaver halves from 53 cadavers, and 5 surgical specimens with a femoral hernia. Graph 1 is the length of the inguinal ligament as measured from the pubic tubercle to the anterior superior iliac spine. Graph 2 is the breadth of the insertion of the posterior inguinal wall (transversus abdominis aponeurosis with fused transversalis fascia) onto Cooper's ligament. Graph 3 is the breadth of the lacunar ligament attachment onto Cooper's ligament. Graph 4 is the transverse diameter of the femoral ring. Graph 5 represents the measurements of the lacunar ligament, the posterior inguinal wall, and the transverse diameter of the femoral ring in 5 cases of femoral hernia. The significant feature here is that only in cases of femoral hernia does the lacunar ligament serve as a medial boundary of the femoral ring, since the lateralmost attachment of the posterior inguinal wall has been pushed medially to correspond exactly to the breadth of the lacunar ligament (see text). The female cadavers are indicated on Graph 2.

so many controversial references available as to the etiology of the femoral hernia this study was undertaken. Utilizing 53 consecutive cadavers of which only 103 cadaver halves were suitable for study, the following measurements were made: The distance between the pubic tubercle and the anterior superior iliac spine (Graph 1, Fig. 1). The breadth of the attachment of the posterior inguinal wall onto Cooper's

ligament (Graph 2, Fig. 1). The breadth of the attachment of the parallel inguinal ligament fibers onto Cooper's ligament, known as the lacunar ligament (Graph 3, Fig. 1). The transverse diameter of the femoral ring as measured from the lateralmost attachment of the posterior inguinal wall onto Cooper's ligament to the medial wall of the external iliac vein (Graph 4, Fig. 1).

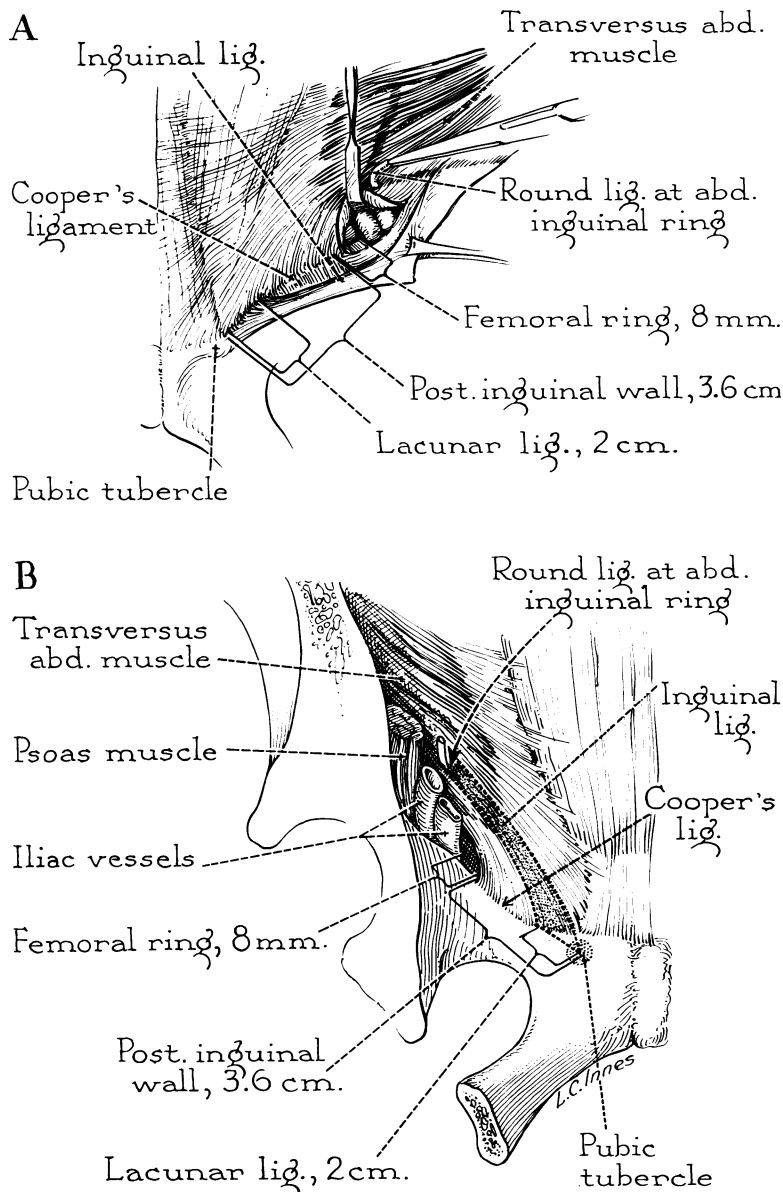


FIG. 2. A. Anterior view of the inguinal region of a female cadaver with the internal oblique and the external oblique removed, leaving only the inguinal ligament and its medial or lacunar attachment. The bracketed measurements show the breadth of the lacunar ligament and posterior inguinal wall attachments onto Cooper's ligament and the transverse diameter of the femoral ring. Note that the medial margin of the femoral ring is not the lacunar ligament in normal anatomy. B. Posterior view of the same specimen with the peritoneum and the preperitoneal connective tissue removed. The same measurements as in the anterior view are bracketed. The inguinal and lacunar ligaments are stippled to show their relative positions and to indicate that they cannot be seen in a posterior view of the inguinal region with an intact posterior inguinal wall. Again note the medial boundary of the femoral ring which is the lateralmost attachment of the posterior inguinal wall onto Cooper's ligament.

Although there were several peritoneal dimples over the position of the femoral ring, in none of these cadavers was there a true femoral hernia. All measurements are exact and stated in centimeters although it must be realized that the transverse measurement of the femoral ring in the cadaver is subject to considerable error. Some of the veins were collapsed and others were over-distended with the injection fluid or a blood clot. The other measurements are exactly accurate because they represent bony prominences and strong aponeurotic attachments to bone.

For comparison, measurements were made on five cases of femoral hernia (Graph 5, Fig. 1). It should be noted that in the case of a femoral hernia, the breadth of the posterior inguinal wall attachment and the breadth of the lacunar ligament attachment are identical. In the first case the transverse diameter of the enlarged femoral ring was less than the transverse diameter of the combined posterior inguinal wall and lacunar ligament attachments. In all others the transverse diameter of the femoral ring was greater.

Figure 2 is the dissection of a female cadaver, drawn accurately to scale and with the various measurements indicated. Figure 2-A is an anterior view of the inguinal region with the external oblique aponeurosis removed except for the inguinal ligament and with the internal oblique muscle and aponeurosis removed. Figure 2-B is a posterior view of the same inguinal region with the peritoneum and preperitoneal connective tissue removed, showing the same structures and with the same measurements appended.

We have made the following point in previous publications,⁴⁻⁷ but it is worthy of re-emphasis. Contrary to what is stated in most textbooks of anatomy, the medial wall of the femoral ring is not the lacunar ligament but is the lateralmost attachment of the posterior inguinal wall onto Cooper's

ligament. The posterior inguinal wall is composed of the transversus abdominus aponeurosis with its fused transversalis fascia. When the femoral ring is dilated to its final limits in the case of a femoral hernia, then the lateral attachment of the posterior inguinal wall is pushed medially until the strong lacunar ligament is encountered. This then represents the final and largest size of the neck of a femoral hernia.

In Figure 2-B, the inguinal and lacunar ligament are outlined by a heavy dotted line and the intervening space is stippled. This is done to show the relative position of the inguinal and lacunar ligaments, and to emphasize that these ligamentous structures cannot be seen in a posterior view of the inguinal region. If the inguinal and lacunar ligaments can be seen in a posterior dissection of the inguinal region, then the posterior inguinal wall has been removed. There are two common sources, one an anatomical atlas and the other a surgical anatomy text which contain a figure showing a posterior view of the inguinal region in which the inguinal and lacunar ligaments are plainly drawn and labeled. These artifactual dissections along with descriptive text in our anatomical text books, perpetuate the idea that the medial margin of the femoral ring is the lacunar ligament; also it would appear that the posterior inguinal wall is inserting onto the inguinal ligament. Operations based upon this premise which attempt to broaden the lacunar ligament attachment to Cooper's ligament and thereby narrow the femoral ring are not only inaccurate but unsatisfactory as attested to by the many devices which have been used in an attempt to permanently broaden the attachment of the lacunar ligament onto Cooper's ligament. A careful study of Figure 2 will not only acquaint one with the normal anatomy of this region but will show the proper method of narrowing the femoral ring in a case of femoral hernia. In other words, the object of

the femoral hernioplasty is to return the anatomy to normal by broadening the attachment of the posterior inguinal wall onto Cooper's ligament so as to obliterate the femoral ring, and it has nothing to do with the inguinal-lacunar ligament system, which is a more superficial structure and rigid in the anteroposterior plane.

Discussion

Figure 1 is a graphic tabulation of the measurements indicated, in a series of cadavers without femoral hernias. This statistical tabulation demonstrates that there is great normal variation not only in the breadth of the lacunar ligament attachment onto Cooper's ligament, but also in the posterior inguinal wall insertion onto Cooper's ligament. There would appear to be no correlation between the breadth of these attachments and the length of the inguinal ligament which is the measurement between the pubic tubercle and anterior superior iliac spine as shown in Graph 1. The measurement of the transverse diameter of the femoral ring is so profoundly affected by the diameter of the external iliac vein, which in the cadaver is so affected by the embalming process that very little reliance can be based upon these measurements (Graph 4).

In studying Figure 1 it will be noted that the measurements frequently appear to go in pairs and this is explained by the fact that the measurements are commonly the same on either side of the same cadaver. On the basis of anatomical probability it would appear that Cases 46 and 47 would be likely to develop a femoral hernia because the posterior inguinal wall insertion onto Cooper's ligament is relatively narrow and approaching the breadth of the lacunar ligament but also the femoral ring is increased in diameter to 1.0 cm. Although Cases 50 and 51 have broad femoral rings, the posterior inguinal wall attachment is relatively broad and probably

does not represent a true dilatation of the femoral ring but rather a collapsed external iliac vein. Case 89, 100 and 101 show the posterior inguinal wall narrowing toward the lateral edge of the lacunar ligament and undoubtedly represent a predisposition to a femoral hernia. A dilated vein could account for the relatively narrow femoral ring. The female cadavers are designated in Graph 2 and there would appear to be no predisposition here for the development of a femoral hernia.

Graph 5, Figure 1, simply shows that in a case of femoral hernia not only is the femoral ring greatly dilated but that the breadth of the attachment of the posterior inguinal wall has been narrowed to correspond exactly to the breadth of the lacunar ligament. If one should question the true nature of the dilated femoral ring in a case of femoral hernia, all that need be done is to cut the inguinal-lacunar ligament (which must be done in some cases) and find that the hernia can still not be reduced because of the constricting femoral sheath.

This anatomic study does not give the complete answer to the etiology of the femoral hernia since there are other factors involved. It does prove, however, that there is great normal variation in the breadth of the attachment of the posterior inguinal wall which may inversely affect the diameter of the femoral ring. A fair statement would be that there is some anatomic predilection to the development of a femoral hernia.

The acquired aspects of the etiology of a femoral hernia have been reviewed in the introduction and of these the lipoma theory would seem to us to be the most tenable. In our experience at the operating table with small femoral hernias, preperitoneal fat is always the presenting portion of the hernia. In eight of 70 cases, preperitoneal fat has been the only constituent of

the hernial bulge, a peritoneal diverticulum being totally wanting.

In considering the etiology of the femoral hernia one cannot ignore the sex incidence or the parous state of the female. Although it has been said that prior to pregnancy the incidence is roughly the same in the two sexes, the parous female in most series has most of the femoral hernias. In our series of groin hernioplasties which now number over 800, we have performed 70 femoral hernioplasties. Analysis of this group of femoral hernioplasties gives some rather startling figures. Contrary to the usual statement on sex incidence, in this group there were 30 males and 40 females. Of the 40 females, 30 had been pregnant one or more times. This leaves only 10 nulliparous females with a femoral hernia. Thus, without the factor of pregnancy, femoral hernia was three times more common in the male than in the nulliparous female. This finding fits in with the statement of Tasche,¹¹ that the available space for the development of a femoral hernia is smaller in the female than in the male.

Some other interesting observations on this group of 70 femoral hernioplasties include the following: in only 11 patients was obesity present; of the eight patients in whom the hernial mass was entirely preperitoneal fat, only three were obese; six patients had bilateral femoral hernioplasties; six of our cases were under ten years of age; and most amazing was the fact that 30 of the 70 cases had associated hernias. The latter statement carries over into our inguinal hernioplasty series⁷ in which the primary diagnosis was an inguinal hernia and a femoral hernia was incidentally discovered, and of course includes the primary femoral hernias in which an unsuspected inguinal hernia was found. This serves to emphasize a point we have made before,⁷ that the "missed" hernia is a common cause of recurrent herniation.

Whereas a strangulated femoral hernia is a rarity in the pregnant female because the hollow viscera are pushed cephalad by the expanding uterus, the increased intra-abdominal pressure secondary to the enlarging uterus must tend to force preperitoneal fat through the femoral ring; or as suggested by Sir Arthur Keith³ the increased pressure within the venous system of the lower extremity in the pregnant female dilates the external iliac vein which in turn stretches the femoral ring, and then fat enters the dilated femoral ring in the postpartum period.

Since the great majority of parous females do not develop a femoral hernia there must be some variation in anatomic structure that predisposes a few of them to the development of a femoral hernia. A congenital diverticulum of peritoneum never exists through the femoral ring and so this cannot be a factor. From the data here presented it would appear that those patients with a relatively narrow attachment of the posterior inguinal wall onto Cooper's ligament and with a correspondingly enlarged femoral ring, are those individuals who are likely to develop a femoral hernia if the factor of sustained increased intra-abdominal pressure is added.

Conclusions

1. The medial margin of the normal femoral ring is the lateralmost attachment of the posterior inguinal wall onto Cooper's ligament and not the lacunar ligament.

2. There is considerable normal variation in the breadth of the attachment of the posterior inguinal wall onto Cooper's ligament.

3. In most instances with a narrow posterior inguinal wall attachment onto Cooper's ligament there is an associated enlarged femoral ring.

4. The primary etiology of the femoral hernia is a congenitally narrow posterior inguinal wall attachment onto Cooper's

ligament with a resultant enlarged femoral ring.

5. The secondary etiology is increased intra-abdominal pressure which forces preperitoneal fat into the congenitally large femoral ring. Pregnancy furnishes a state of prolonged and sustained intra-abdominal pressure which is the common secondary cause of a femoral hernia.

Acknowledgment

In addition to our own cadavers at the University of South Dakota, we examined the cadavers at Northwestern University Medical School and the University of Chicago with the kind permission of the chairman of these Departments of Anatomy.

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